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Original Article

# Median and Phrenic Nerve Conduction Study in Patients with Type II Diabetes Mellitus

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## Abstract

**Background:** Diabetes mellitus is an iceberg disease. Unfavorable modification of lifestyle and dietary habits that are associated with urbanization is believed to be the most important factors for the development of diabetes. Diabetic patients, if undiagnosed or inadequately treated, develop multiple chronic complications leading to irreversible disability and death.

**Aim and Objective:** The aim of this study is to compare the motor median and phrenic nerve conduction study in Type II diabetic patients.

**Materials and Methods:** Forty-five diabetic patients were recruited and were subjected to do median and phrenic nerve conduction study. Results were statistically analyzed by ANOVA.

**Results:** There was a significant ($P < 0.05$) increase in latency and decrease in amplitude and nerve conduction velocity of both phrenic and median nerve conduction.

**Conclusion:** We conclude that like other peripheral nerves phrenic nerve also gets affected in Type II diabetes mellitus.

**Key words:** Amplitude, Diabetes mellitus, Latency, Median nerve, Phrenic nerve

## INTRODUCTION

Diabetes mellitus is characterized by chronic hyperglycemia with disturbances of carbohydrate, fat, and protein metabolism, resulting from defects in insulin secretion, insulin action, or both. Type II diabetes is the most common form of diabetes. Patients with Type II diabetes usually have insulin resistance, rather than absolute, insulin deficiency. Their circulating insulin levels may be normal or elevated yet insufficient to control blood glucose levels within the normal range because of their insulin resistance. The effects of diabetes mellitus include long-term damage and dysfunction of various organs, especially the eyes, kidneys, heart, and blood vessels.[1]

Diabetic neuropathy encompasses a wide, heterogeneous group of clinical and subclinical syndromes. It is one of the major long-term complications associated with diabetes that can cause considerable morbidity and mortality.[2] 50–75% of all ulcerations and non-trauma amputations are a consequence of diabetic neuropathy, and it causes more hospitalizations than all the other diabetic complications.[3,4] Diabetic neuropathy affects the sensory, autonomic, and motor neurons of the peripheral nervous system.[5] Neuropathy generally progresses at a steady state given that the level of impairment directly correlates with the duration of diabetes.[5]

The diaphragm, principal inspiratory muscle is supplied by the phrenic nerve. Phrenic nerve arises from the 3rd, 4th, and 5th cervical segment of the spinal cord. It is a mixed nerve having both sensory and motor component. Diaphragm weakness implies a decrease in the strength of the diaphragm. Diaphragm paralysis is the extreme form of diaphragm weakness. Weakness of the diaphragm most frequently arises from diseases in the phrenic nerve or from myopathies affecting the diaphragm secondary to some disease process.[6]

Reduced muscle strength has been reported in diabetic patients. Bilateral or unilateral diaphragmatic paralysis

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has been observed in diabetic patients.[7] In a study, they had reported that the respiratory muscle endurance was impaired, and a greater perception of respiratory exertion was noticed in diabetic patients relative to their matched controls.[8] Moreover, breathlessness on exertion and orthopnea in association with Type II diabetes mellitus has been also reported. Investigation showed that bilateral diaphragmatic paralysis due to phrenic neuropathy may be an important, if rare complication of diabetes and diaphragmatic function should be considered in any patient with unexplained breathlessness and orthopnea.[9]

Hence, this study is aimed to detect the nerve conduction parameter of phrenic nerve and median nerve in Type II diabetes mellitus patients.

## MATERIALS AND METHODS

It is a hospital-based cross-sectional study which was done in the Physiology Department of Sree Balaji Medical College Hospital and Research Institution, Chennai. We selected 45 Type II diabetic patients with the age group of 35–55 years. Among them, 15 were males and 30 were females. Ethical Committee clearance was obtained. Well-informed written consent was obtained from all those who participated in the study. We excluded the patients suffering from thyroid diseases, renal disorders, and liver diseases. Based on the duration of diabetes mellitus, all the patients involved in the study were divided into three groups. With <5 years of duration of diabetes belong to one group, 5–10 years of duration in other group, and >10 years of duration in one another group.

The fasting, postprandial blood sugar (PPBS) values, and glycosylated levels of the patients were assessed. All the patients were subjected to do motor median and phrenic nerve conduction study in the research laboratory of the physiology department. All the results were statistically analyzed and tabulated. Statistical analysis was performed using ANOVA.

## DISCUSSION

All our patients involved in the study, in spite of having regular medication, were suffering from higher mean values of fasting, PPBS level, and glycosylated hemoglobin (HbA1c), which was also found to be correlated with duration of diabetes [Table 1 and Figures 1 and 2]. The motor median nerve conduction study of both right and left side of all diabetic patients showed an increase in mean latency as the disease progressed [Tables 2 and 3]. Whereas, the amplitude and motor nerve conduction velocity were decreased as the duration of disease increased. This study is in par with the study done by Lewko *et al*.,[10] who also had an increase in nerve conduction velocities as the disease progressed.

Similarly, the nerve conduction parameters of the phrenic nerve also showed almost the same result as that of median nerve. The nerve conduction velocity of phrenic nerve was reduced on both sides. The latency of the nerve conduction increased, whereas the amplitude of the nerve conduction reduced on both sides [Table 4 and Figures 3 and 4]. Our study is in par with the study of Bansal *et al*.,[11] who had suggested that the slowing of nerve conduction velocity indicates the ongoing damage to the myelin sheaths and concluded that nerve conduction velocity is gradually diminished in diabetic neuropathy.

Amplitude reflects the size and number of nerve fibers, and its measurement is important for the evaluation of neuropathy. Both latency and conduction velocity depend on an intact, myelinated nerve as myelin and the saltatory conduction are essential for fast action potential propagation in normal subjects. Slowing of conduction velocity or prolongation of latency usually implies demyelinating injury, while the loss of amplitude usually correlates with axonal loss or dysfunction.[12]

Thus, our study proves that like other peripheral nerves, phrenic nerve also gets affected in Type II diabetes mellitus. The reason for increase in latency and decrease in amplitude of both phrenic and median nerve is due to nerve damage caused by hyperglycemia. The pathophysiology of diabetic neuropathy includes increased oxidative stress yielding advanced glycosylated end products, polyol accumulation, decreased nitric oxide/impaired endothelial function, and impaired Na+/K-ATPase activity. Hyperglycemia not only causes mere destruction to nerve fibers but also the repair mechanisms are also defective.[13-15]

Unilateral paralysis might not only present clinically but also bilateral paralysis causes respiratory failure and might end in mortality. Phrenic neuropathy should be considered

**Table 1: Basic characteristics of all the diabetic patients**

| Duration | <5 years (*n*=15) | 5–10 years (*n*=15) | >10 years (*n*=15) |
|---|---|---|---|
| Age (years) | 42.6±5.6 | 48.6±4.3 | 53±2.7 |
| Height (cm) | 152.5±9.5 | 150.5±5.8 | 154.2±8.1 |
| Weight (Kg) | 60±7.1 | 62±8.3 | 63±6.4 |
| BMI (Kg/m²) | 25.2±4.4 | 27.6±4.3 | 26.5±2.8 |
| FBS (mg/dl) | 112.5±20* | 137.5±34.6* | 151.4±47.8* |
| PPBS (mg/dl) | 197.3±48* | 215.6±44.2* | 218.5±53* |
| HbA1c (%) | 7.21±0.8 | 7.39±0.9 | 7.52±1.3 |

FBS: Fasting blood sugar, PPBS: Postprandial blood sugar, BMI: Body mass index, HbA1c: Glycosylated hemoglobin. *Significant (*P*<0.05)

**Table 2: Right side motor median nerve conduction study of all diabetic patients**

| Duration (years) | Latency (ms) | | Amplitude (mv) | | Latency diff (ms) | Conduction velocity (ms) |
|---|---|---|---|---|---|---|
| | Wrist | Elbow | Wrist | Elbow | | |
| <5 | 2.91±0.66 | 7.1±0.6 | 5.8±3.9 | 4.7±4.1 | 4.21±0.38 | 49.9±5.5* |
| 5–10 | 3.1±0.6 | 7.5±0.9 | 6±3.3 | 4.7±2.4 | 4.4±0.5 | 48.7±5.1* |
| >10 | 4.5±1.8 | 9.04±2 | 2.48±2.4 | 2.19±2.4 | 4.5±0.3 | 47.9±3.6* |

*Significant ($P$<0.05)

**Table 3: Left side motor median nerve conduction study of all diabetic patients**

| Duration (years) | Latency (ms) | | Amplitude (mv) | | Latency diff (ms) | Conduction velocity (ms) |
|---|---|---|---|---|---|---|
| | Wrist | Elbow | Wrist | Elbow | | |
| <5 | 2.8±0.7 | 7.1±0.9 | 6.04±3.3 | 4.7±4.1 | 4.26±0.7 | 49.3±5.9* |
| 5–10 | 3.39±1.25 | 7.67±1.3 | 6.3±3.9 | 4.3±1.9 | 4.28±0.5 | 48.3±4.7* |
| >10 | 4.1±2.5 | 8.2±1.3 | 4.29±2.5 | 3.67±2.2 | 4.6±0.6 | 45.4±6.08* |

*Significant ($P$<0.05)

**Table 4: Motor phrenic nerve conduction study of all diabetic patients**

| Parameters (years) | Latency (ms) | | Amplitude (mv) | | Nerve conduction velocity (ms) | |
|---|---|---|---|---|---|---|
| | Right | Left | Right | Left | Right | Left |
| <5 | 10.3±4* | 9.4±2.8* | 0.5±0.6 | 0.52±0.3 | 3.4±1.2** | 3.7±1.5** |
| 5–10 | 12±2.4* | 10.2±3.1* | 0.45±0.4 | 0.48±0.5 | 2.4±0.8** | 3±0.6** |
| >10 | 12.7±5.1* | 12.6±4.7* | 0.4±0.3 | 0.32±0.3 | 2.2±0.6** | 2.8±1.3** |

**Highly significant ($P$<0.001). *Significant ($P$<0.05)

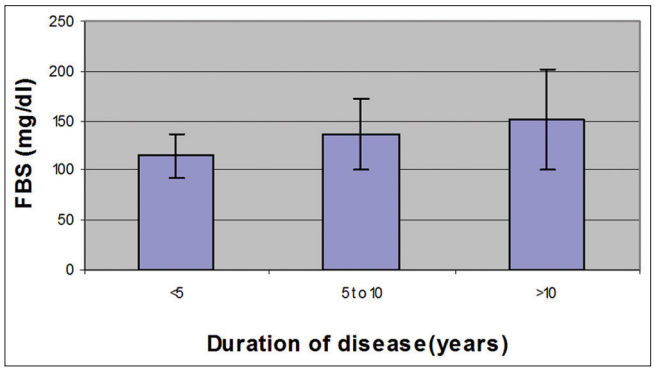


**Figure 1: Comparison between fasting blood sugar and duration of disease**

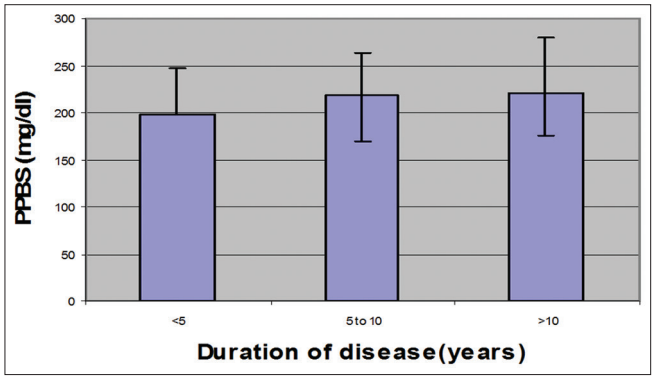


**Figure 2: Comparison between postprandial blood sugar and duration of disease**

as an important complication of diabetes. Phrenic nerve conduction should be assessed routinely like other peripheral nerves to prevent morbidity and mortality.

## RESULTS

Table 1 presents the basic physical characters of all the diabetic patients as age, height, weight, body mass index, and the blood parameters as fasting blood sugar, PPBS, and HbA1c. It is clearly evident that as the duration progresses the blood glucose levels are not under control.

Table 4 summarizes the motor phrenic nerve conduction study of both sides. It is obvious that latency increases (statistically significant) and amplitude decreases with the progression of duration of illness. The nerve conduction velocity is significantly reduced on both sides.

Tables 2 and 3 present the motor median nerve conduction study results among the diabetic patients. Here, also the latency increases, whereas the amplitude decreases. The nerve conduction velocity is also significantly reduced in both sides. Figures 1 and 2 show the glycemic status in diabetic patients of varying duration.

Figures 1 and 2 represent the bar diagram of the glycemic indices, namely, fasting and PPBS and their comparison

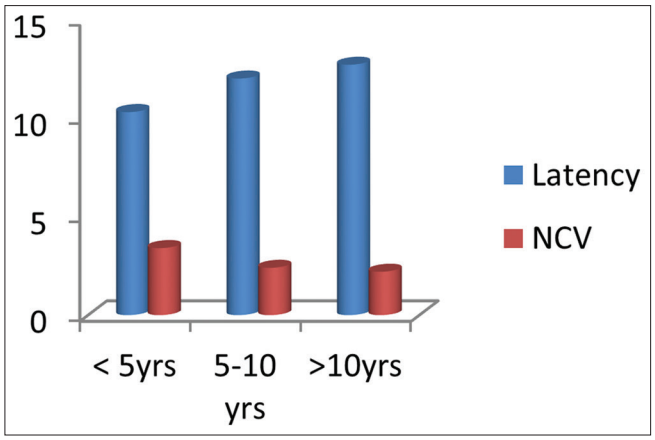


**Figure 3: The bar diagram of the right side motor nerve conduction of phrenic nerve**

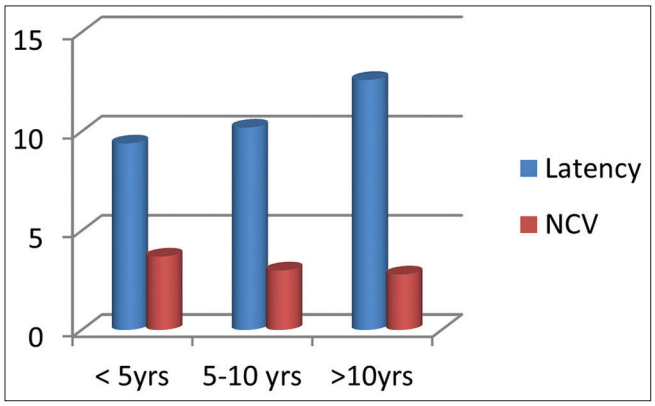


**Figure 4: The bar diagram of the left side motor nerve conduction of phrenic nerve**

with duration of disease. It can be clearly observed that as duration progresses the glycemic status worsen.

Figures 3 and 4 represent the bar diagram of the right and left side motor nerve conduction of phrenic nerve, respectively. It is obvious that as the duration of the disease progresses the latency increases and conduction velocity decreases.

## CONCLUSION

We conclude that like other peripheral nerves phrenic nerve also gets affected in Type II diabetes mellitus.

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